IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
No. 3:25-cv-00199-JDA

MARION BOWMAN, JR., Plaintiff, v. BRYAN P. STIRLING, in his official capacity as the Director of the South Carolina Department of Corrections, and; HENRY DARGAN McMASTER, in his official capacity as Governor of the State of South of South Carolina, Defendants.	**CAPITAL CASE** **EXECUTION SCHEDULED JANUARY 31, 2025**

DECLARATION OF JOSEPH F. ANTOGNINI, M.D., M.B.A.

JOSEPH F. ANTOGNINI, does hereby declare and say:

1. My name is Joseph F. Antognini. I am a medical doctor, board-certified in anesthesiology. I received a B.A. degree from the University of California, Berkeley in Economics in 1980. I received my M.D. degree from the University of Southern California in 1984. I also received an M.B.A. from California State University, Sacramento in 2010. I was previously the Director of Peri-operative Services at the University of California, Davis Health System and a Professor of Anesthesiology and Pain Medicine and Professor of Neurobiology, Physiology and Behavior at the University of California, Davis. I am licensed to practice medicine in the State of

California. I have over 30 years of experience practicing anesthesiology since 1984 when I began my residency at the University of California, Davis Health System. I am the author or co-author of over 200 publications (papers, abstracts, book chapters, etc.). My area of research has focused on anesthetic mechanisms, specifically related to where anesthetics produce unconsciousness, amnesia and immobility. I currently perform clinical research, and I am Chief Scientific Officer for a small pharmaceutical company that develops new anesthetics. A true and correct copy of my curriculum vitae is attached hereto as Exhibit A.

2. I have reviewed, and am familiar with, the allegations made in the Motion for Injunctive Relief, *Marion Bowman, Jr. v. Bryan P. Stirling and Henry Dargan McMaster*, No. 3:25-cv-00199-JDA, dated January 13, 2025, and additional information in the documents described below.

Scope of Engagement

3. I have been asked to render expert opinions in the fields of general medicine and anesthesiology, especially regarding the use, actions and efficacy of pentobarbital, in relation to South Carolina's lethal injection protocol, and the effectiveness of the procedures therein. This declaration contains a complete statement of my opinions, and the basis and reasons therefore, including the facts or data I have considered in forming them. I may supplement this declaration as appropriate. The opinions that I do

provide are within my field of anesthesiology and such fields as are necessarily related to anesthesiology, including general medicine, pharmacology and physiology, and fall within the scope of my expertise. All opinions expressed herein are stated to a reasonable degree of medical and scientific certainty unless otherwise noted.

Materials Reviewed

4. I have conferred with attorneys for Defendants. Among the documents I have reviewed in connection with this case are: South Carolina's execution protocol; Plaintiff's Motion for Injunctive Relief (dated January 13, 2025); publications and materials listed in the "References Cited" section; the declaration of Dr. David Waisel (dated January 10, 2025); the affidavit of Bryan P. Stirling, dated January 7, 2025; the affidavit of Dr. Michaela Almgren, dated August 31, 2024; and the autopsy report on Richard Moore.

5. Should additional documents or information be provided to me for review and analysis, I may take those additional materials into account, and modify and/or supplement my opinions accordingly. If I am present at hearings and/or trial in this case, I may take into account any testimony or other evidence to the extent related to my opinions and modify and/or supplement my opinions accordingly. In performing my analysis, I have relied on my professional training, education and experience. The opinions

presented in this declaration are my opinions and mine alone. I have reviewed and considered documents and information and identified those materials above. These documents and other information that I reviewed and considered are of a type reasonably relied upon by experts in the field of anesthesiology, general medicine, physiology and pharmacology in forming opinions or inferences on questions in this area. My fee schedule for this engagement is: $575/hour for phone consultation, research, declaration preparation; $675/hour for deposition; $7000/day for courtroom appearance; $287/hour for travel time plus travel expenses at cost.

6. I have testified and submitted expert reports in the following cases in the past four years: 1) I have submitted reports and given testimony *In the Matter of the Federal Bureau of Prisons' Execution Protocol Cases* (No. 19-mc-00145-TSC); 2) I have submitted reports and have testified in *Glossip et al. v. Chandler et al.*, Case No. CIV-14-665-F, in the United States District Court for the Western District of Oklahoma; 3) I have submitted reports and have testified in *Bigler Stouffer. v. Scott Crow*, Case No. 21-cv-1000-F, in the United States District Court for the Western District of Oklahoma; 4) I have submitted reports and have been deposed in *Terry Lynn King v. Tony Parker*, Case No. 3:18-cv-01234, in the United States District Court for the Middle District of Tennessee; 5) I have submitted reports and testified in *Michael Nance v. Oliver & Caldwell,* Case No. 1:20-CV-107-JPB, in the United States

District Court for the Northern District of Georgia, Atlanta Division; 6) I have submitted reports and testified in *Kenneth Eugene Smith v John Q. Hamm*, 2:22-cv-00497-RAH, in the United States District Court for the Middle District of Alabama; 7) I have submitted reports and been deposed in *Martin v Oliver & Caldwell,* 1:18-cv-4615-MLB in the US District Court, Northern District of Georgia, Atlanta Division; 8) I have submitted a report and been deposed in *Miller v. Marshall et al.* 2:24-cv-197 in the United States District Court for the Middle District of Alabama; 9) I have submitted a report and testified in *Grayson v. Hamm et al.,* 2:24-cv-00376-RAH-KFP in the United States District Court for the Middle District of Alabama.

Discussion

7. The intravenous administration of five (5) grams of pentobarbital causes rapid unconsciousness followed by respiratory arrest, cardiovascular collapse and death. After intravenous injection of 5 grams pentobarbital, concentrations of pentobarbital in the body will far exceed the lethal concentrations—see Table 1, package insert for pentobarbital in References Cited and extrapolating from data of Ehrnebo (1974). Once respiratory depression and respiratory arrest occurs within 1-2 minutes, the unconscious inmate then begins to use up the oxygen stores in his body. Before all the oxygen is used, however, the heart will be affected, will begin to slow and will then have periodic irregular beats. It likely will take several minutes before

the heart stops all together. At that point, death is declared. This process, as described, is irrefutable. It is based on the known actions of pentobarbital and sound pharmacological and physiological principles, and the known effects of these doses of pentobarbital in lethal injection executions.

8. Pentobarbital administered to humans results in unconsciousness in 20-30 sec, on average,[1] and this effect is dose dependent, with greater doses (>5 mg/kg) having onset times in the 20 sec range (Dundee, 1957). In a 100-kg person (about 220 pounds), this dose would be 500 mg, which is only 10% of the dose used in the South Carolina lethal injection protocol. At this point, pulmonary edema, if it occurs at all during the execution (as opposed to post-mortem lung changes), would not set in because it would only result from a much larger dosage (i.e. an overdose).[2] As the additional 4500 mg of pentobarbital is administered, the inmate would have progressive brain depression, with electrical brain silence occurring, followed by cardiovascular collapse, as noted above. Before becoming unconscious, the individual would not feel the sensations of pain, suffocation or air hunger. And the inmate

1 It is important to note that the time to unconsciousness depends on the speed with which the drug is administered and when the "clock starts". For example, my estimate for 20-30 sec is based on when a clinical dose of pentobarbital has actually entered the person, not when the drug begins to enter the IV tubing.
2 Clinical doses of barbiturates, such as thiopental and pentobarbital, cause unconsciousness, but not pulmonary edema. If clinical doses of these drugs caused pulmonary edema, the drugs would have been abandoned soon after their introduction in the 1930s.

would not feel the sensation of pain, suffocation or air hunger after becoming unconscious.

9. These actions of pentobarbital are consistent with data published by Aleman et al., (2015), a study discussed in the recent US Supreme Court case *Bucklew v. Precythe*, No. 17-8151 (decided April 1, 2019). In the Aleman study, horses were administered large, lethal doses of pentobarbital, with a mean time of infusion of 47 seconds, and the horses developed electroencephalographic brain silence (i.e., flat line) at a mean of 53 seconds after the initiation of the infusion, that is, EEG silence occurred on average, 6 seconds after the infusion finished. Because loss of consciousness occurs before EEG silence, these data fit with a time frame of 20-30 seconds for loss of consciousness after the initiation of the pentobarbital infusion.

10. In a similar study (Buhl et al., 2013), the time to collapse (when the horses went from standing to falling to the ground, and which is considered to be the onset of unconsciousness) was about 27 seconds (the average of the means of the four groups studied; see their table 2) after the initiation of the infusions. They also noted that respiratory arrest occurred simultaneously with falling to the ground in most horses (2nd paragraph in discussion).

11. These studies cited above collectively lead to the conclusion that intravenous pentobarbital administered at 5 grams would cause rapid onset

of unconsciousness, followed by coma, respiratory arrest, circulatory collapse and death.

12. Thiopental and pentobarbital are equipotent (Barron & Dundee, 1961). For example, 100 mg of thiopental has the same effect as 100 mg of pentobarbital, 500 mg of thiopental has the same effect as 500 mg of pentobarbital, and so forth. Thus, studies reporting on the effects of thiopental can be used to infer the effects of pentobarbital.

13. Both thiopental and pentobarbital cause brain suppression (including suppression of electrical activity in the brain as measured with the electroencephalogram, EEG). The dose at which EEG silence begins to occur is about 17 mg/kg, based on studies utilizing thiopental infused over 10-15 minutes (Buhrer et al., 1992; Hung et al., 1992). But, in the setting of an execution, pentobarbital would be infused more quickly and at a greater dose than that described in Buhrer et al. Five (5) grams (equivalent to 5000 mg) of pentobarbital administered to a 100-kg person (approximately 220-lbs person) is 50 mg/kg, and about 71 mg/kg in a 70 kg person, doses that far exceed 17 mg/kg. Thus, EEG silence would be expected to occur within 60 seconds after initiation of pentobarbital infusion, consistent with the data reported by Aleman et al.

14. The State of Georgia has executed at least 24 inmates in the past decade using pentobarbital, and these times to death were submitted as evidence in *Martin v. Ward & Ford*, No. 1:18-cv-04617-MLB, in which both Dr Waisel and I were expert witnesses.[3] The times between initiation of pentobarbital infusion and time of death reported for 24 executions ranged from 8 to 27 minutes, with an average of about 14 minutes. These times comport with what I would expect with 5 grams of pentobarbital administered according to the South Carolina protocol. The longer times between pentobarbital administration and time of death are most likely related to the process by which death is declared related to cessation of electrical heart activity. The electrocardiogram (ECG) measures electrical heart activity and in the process of dying the heart may have occasional electrical activity for many minutes after complete cardiovascular collapse and respiratory arrest. The amount of time it takes for the heart to stop can be variable, so the ranges reported for these 24 executions are not surprising and do not indicate any problems with the way in which the Georgia protocol is implemented.[4,5]

3 Dr. Waisel considered and discussed these times in his May 10, 2024 report submitted to the Court, as did I in my report dated April 10, 2024.

4 Also, during an execution there is an additional variable amount of time between when the heart stops beating and when time of death is determined. The physician must wait a variable amount of time after the last heartbeat to ensure he or she has actually observed the "last" heartbeat. This time might be 1-2 minutes or longer, depending on the physician. Then, the physician enters the chamber, examines the inmate for signs of life, and declares the time of death.

5 The Georgia method of lethal injection execution and the South Carolina method are similar

15. In the Aleman study, horses administered pentobarbital developed asystole (cessation of the heartbeat) in the range of 5.5 to 16.3 minutes (a ratio 16.3/5.5 ≈ 3), and in the Buhl study the range was 3.3 to 20 minutes (based on the data in their Figure 2; ratio 20/3.3 ≈ 6). The ratio of the times to death (longest/shortest) in the 24 executions is 27/8 ≈ 3.4, like those found in the Aleman and Buhl studies. Taken together, these execution times and the animal studies indicate that variability is the norm, not the exception.

16. Intravenous administration of 5 grams of pentobarbital would cause profound brain depression and unconsciousness well before any lung congestion and pulmonary edema forms.

17. Whether pentobarbital causes pulmonary edema directly, or indirectly as a natural consequence of the dying process, is immaterial because the inmate would be profoundly unconscious, to the point of electrical brain silence. Furthermore, it is unclear how much of the pulmonary edema and lung congestion found at autopsy is due to post-mortem changes.

18. More recent studies in humans using post-mortem computed tomography (PMCT) show that fluid accumulates in lung over time in the post-mortem period (Shiotani et al., 2011). Shiotani et al. write in their concluding paragraph: “PMCT findings of the lung are not fixed and change

regarding procedures and administration of pentobarbital.

with the passage of time after death in accordance with progression of postmortem changes (pulmonary congestion and edema) in the corpse."

19. Likewise, fluid accumulation in the airways increases during the post-mortem period (Ishida et al. 2014); these authors showed that fluid accumulated in the airways (main bronchi) as the interval between death and PMCT increased. This fluid accumulation is akin to the fluid that has been found at autopsy in inmates executed by lethal injection.

20. Published data on how post-mortem pulmonary edema and lung congestion occur and progress is based in large part on animal studies. Durlacher et al. (1950) examined post-mortem changes in rabbit lungs after various causes of death, including pentobarbital overdose. They found that lung weight increased as the time between pentobarbital-induced death and autopsy increased, as shown in their table 2:

TABLE 2

EFFECT OF INTERVAL AFTER SACRIFICE BY NEMBUTAL (100 MG./KG.) ON LUNG WEIGHT

Interval after sacrifice	*Treatment*	*Number of animals*	*Lung weight per kilo ± S.E. mean*
			Grams
Immediate		5	3.83 ± .27
1 hours	Cannula in trachea	5	5.42 ± .58
2 hours	Cannula in trachea	5	7.09 ± 1.39
3 hours	Cannula in trachea	19	9.46 ± .62
4 hours	Cannula in trachea	5	10.88 ± 1.53
6 hours	Cannula in trachea	5	10.95 ± .74

Note that lung weight increased when comparing lung weight at immediate autopsy to lung weight at 1, 2, 3, 4 and 6 hours after death, indicating that

lungs can develop edema *after* death. These researchers (and others[6]) also found that, for a variety of causes of death, lung weight increased as the interval between death and autopsy increased (see table 1 in Durlacher et al., 1950). These data indicate that post-mortem edema formation is a generalized phenomenon and is not specific to drug overdose Thus, the animal data indicate that all of the pulmonary edema and lung congestion found at autopsy in inmates executed by lethal injection could be generated post-mortem.

21. Frothy fluid and foam are sometimes found in humans and animals after death, and there is evidence that this froth can occur immediately prior to death (in the period from apnea to cardiac death; see Swann 1964) and after death.[7] Thus, the finding of froth in inmates who were executed by lethal injection does not indicate that this froth was generated ante-mortem.

22. Post-mortem froth and foam could be generated by the release of gasses from the lung tissues and interacting with the lung surfactant, a substance that, during life, keeps alveoli (small lung units, or air sacs) open. Related to this issue, Pattle (1955) wrote that "….oedema foam is thus not produced by

[6] See Acta Scandinavica Medica 1964 in References Cited

[7] Animals (rabbits) made uremic (kidney failure) and who subsequently developed pulmonary edema were found to not only have increasing lung weights as the period between death and post-mortem exam increased, but the presence of froth was found in animals that had later post-mortem exams, while none was found upon immediate post-mortem examination. See Acta Scandinavica Medica 1964 in References Cited

agitation of the oedema fluid with air during respiration; it can only have been formed by air originally in the fine air spaces of the lung being broken up into bubbles and afterwards expelled into the bronchi and trachea." Thus, the post-mortem finding of froth in inmates who were executed by lethal injection does not conclusively indicate that this froth was generated ante-mortem, or by conscious attempts to breathe.

23. The presence of pulmonary edema at autopsy is a common and non-specific finding and is associated with variety causes of death (Saukko & Knight, 2004; Sogawa et al., 2014).[8]

24. The witnesses to the executions of Freddie Owens and Richard Moore describe what would be expected from lethal injection of pentobarbital (see links to the press conferences cited in the References section). In the Owens execution, Owens appeared to be conscious for about 1-1.5 minutes after initiation of the pentobarbital, followed by deep breathing akin to snoring, then shallow breathing. No movement occurred after about 6 minutes following the initiation of the pentobarbital. In the Richard Moore execution, several deep breaths started about 1 minute following the initiation of the pentobarbital, followed by shallow breathing, with no movement observed

8 Saukko & Knight: Knight's Forensic Pathology, 3rd Edition, page 356: "Pulmonary oedema is such a common and non-specific phenomenon in a whole range of fatal conditions that it has little diagnostic significance."

after about 3 minutes. These observations comport with what I would expect to occur following a lethal dose of pentobarbital.

25. In his declaration Dr. Waisel lists several opinions that are not well-founded and are based on faulty reasoning and erroneous interpretation of the data and events.[9] In section V.7 of his declaration, he opines that a properly administered dose of pentobarbital should eliminate breathing in less than one minute, but he ignores important factors. For example, the speed with which the drug is administered impacts responses to the drug, with slower administration causing longer times for drug effects to occur. Also, the presence of agonal breaths, which are the last "gasps" that a person or animal takes immediately prior to death, prolongs the time to apnea (lack of breathing). In my opinion, breathing efforts can occur for a few minutes after the pentobarbital has been administered, however, these breathing efforts will become shallow in the few minutes after drug administration.

26. In section V.8 Dr. Waisel states that it is "physiologically and pharmacologically impossible for Mr. Moore to remain alive for ten minutes after a dose of five grams of fully-potent pentobarbital, unless that dose was not delivered completely". Dr. Waisel completely ignores the expected effect of

9 As an aside, Dr. Waisel discusses in section V.2 of his declaration the hypothetical administration of a barbiturate at a dose of 350 mg/kg, which is clearly a typographical error. But because the reader is left wondering what dose he meant to write, Dr. Waisel's reasoning is further muddled.

the pentobarbital and the practical aspects of the execution process. As outlined above, the heart can have occasional beats for many minutes after pentobarbital is administered (as long as 20 minutes between drug administration and loss of cardiac electrical activity—see Buhl et al., 2013). While the inmate is deeply unconscious, the person who declares death will not do so until a waiting period after the electrocardiogram is "flatline", e.g., there is no electrical activity of the heart.

27. In section V.9, Dr. Waisel opines that Mr. Moore "consciously experienced feelings of drowning and suffocation during the 23 minutes that it took to bring about his death". Dr. Waisel completely ignores the effects of the lethal doses of pentobarbital used. Dr. Waisel expects the reader to believe that the massive dose of pentobarbital will not cause unconsciousness but will result in "sudden" death at minute 23. If Mr. Moore was conscious and drowning in his own fluids then why didn't he move prior to minute 23? Why didn't he breathe fast, as would be expected if he was awake and had pulmonary edema? The answer to both questions is that Mr. Moore was profoundly unconscious from the pentobarbital.

28. Dr. Waisel also questions the need for an additional 5-grams of pentobarbital in the Moore execution. As noted above, it would not be unexpected that some electrical activity of the heart persisted after 10

minutes, so a second 5-gram injection was probably used for that reason. The witnesses to the Moore execution reported that Mr. Moore did not move after about 3 minutes, so Mr. Moore likely was profoundly unconscious at that point. Also, the pentobarbital concentration found in Mr. Moore at autopsy (85 mcg/ml) greatly exceeded the lethal level.[10] In the 24 Georgia executions using 5-grams pentobarbital, the mean pentobarbital concentration at autopsy was 38 mcg/ml, so the 85 mcg/ml level found in Mr. Moore is consistent with the administration of a 10-gram dose of pentobarbital, which refutes Dr. Waisel's claim that an insufficient amount of pentobarbital was administered.[11]

29. In section V.11 Dr. Waisel states that intravenous access in obese persons might be difficult, but this is true for any patient, and thousands of obese patients have surgery every day after the successful placement of an intravenous catheter.

Conclusion

30. It is my opinion, to a reasonable degree of medical and scientific certainty, that 1) the inmate would become unconscious within 20-30 sec after pentobarbital first enters the inmate, which would be followed by respiratory arrest, cardiovascular collapse and death; 2) injection of massive

10 The package insert states that 10-15 mcg/ml causes coma, while 15-40 mcg/ml is lethal.
11 In his report of May 10, 2024, Dr. Waisel states he reviewed the toxicology data for these executions.

doses (5 grams) of pentobarbital would not inflict mild, moderate or severe pain; 3) pulmonary edema, if it occurs ante-mortem, would not be perceived by the inmate because of the profound brain suppression caused by pentobarbital.

31. Should additional information become available I reserve the opportunity to amend my statements herein.

Date: January 21, 2025

Joseph F. Antognini, M.D., M.B.A.

References Cited

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Shiotani S, Kobayashi T, Hayakawa H, Kikuchi K, Kohno M. Postmortem pulmonary edema: A comparison between immediate and delayed postmortem computed tomography. Legal Medicine 2011; 13:151-55

Sogawa et al., Postmortem virtual volumetry of the heart and lung in situ using CT data for investigating terminal cardiopulmonary pathophysiology in forensic autopsy. Legal Medicine 2014;16:187-92

Swann HE. The development of pulmonary edema during the agonal period of sudden asphyxia deaths. J Forensic Sciences 1964; 9:360-73

Press conference following execution of Richard Moore on November 1, 2024 (accessed January 18, 2025):

Witnesses speak after execution of South Carolina inmate Richard Moore

Press conference following execution of Freddie Owens on September 20, 2024 (accessed January 18, 2025):

FULL PRESS CONFERENCE Freddie Owens Execution: 9.20.2024

Pentobarbital package insert (accessed 1-21-2025):

https://dailymed.nlm.nih.gov/dailymed/fda/fdaDrugXsl.cfm?setid=e9f4b344-

b092-4eec-b49d-d8cfe8ebc05d&type=display

EXHIBIT A

CURRICULUM VITAE
Joseph F. Antognini, M.D., M.B.A.

CONTACT:

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EDUCATION:

1980	University of California, Berkeley (B.A., Economics)
1984	University of Southern California (M.D., Medicine)
2010	California State University, Sacramento (M.B.A., Business)

INTERNSHIP/RESIDENCY:

1984-1987	Anesthesiology, UC Davis Medical Center
1986-1987	Chief Resident

PROFESSIONAL POSITIONS:

6/24-present	Chief Scientific Officer/Interim Chief Medical Officer Expanesthetics, Inc Davis, CA
1/22-present	Principal Investigator Next Level Clinical Trials, LLC West Covina, CA
1/22-present	Sub-Investigator SmartCures Clinical Research, LLC Anaheim, CA
7/22-present	Sub-Investigator Long Beach Clinical Trials, LLC Long Beach, CA

7/17-present	Director Emeritus University of California, Davis
2015-present	Clinical Advisory Board Expanesthetics, Davis, CA
9/21-7/23	Surgical Wound Specialist Advantage Surgical and Wound Care El Segundo, CA
1/20-12/22	Adjunct Faculty Los Medanos College Pittsburg, CA
1/20-5/20	Adjunct Faculty Holy Names University Oakland, CA
9/16-11/19	Physician Surveyor The Joint Commission Oakbrook Terrace, IL
2011-2020	Clinical Professor of Anesthesiology and Pain Medicine (Volunteer Clinical Faculty appointment) University of California, Davis—School of Medicine
11/10-6/16	Director of Peri-operative Services UC Davis Health System
7/00-7/11	Professor of Anesthesiology and Pain Medicine12 (with tenure) Department of Anesthesiology and Pain Medicine University of California, Davis—School of Medicine
12/02-7/11	Professor of Neurobiology, Physiology and Behavior (with tenure; WOS appointment) College of Biological Sciences

12 My research publications place me in the top 1.5% of scientists worldwide based on number of citations of my papers (October 2023 data-update for "Updated science-wide author databases of standardized citation indicators" - Elsevier BV (digitalcommonsdata.com) accessed 5-17-2024). Also, I am in the category of “outstanding scientist” based on the h-index (h-index = 42 as of 1-9-25, with >5600 citations according to Google Scholar). The h-index is a measure of how often a person’s work is cited. See: Hirsch JE. An index to quantify an individual’s scientific output. PNAS 2005; 103:16569-572

	University of California, Davis
11/98-7/10	Vice Chairman, Director of Research
11/98-3/02	Director of Malignant Hyperthermia Diagnostic Laboratory Department of Anesthesiology
7/96-7/00	Associate Professor (with tenure) Department of Anesthesiology University of California, Davis—School of Medicine
10/91-6/96	Assistant Professor Department of Anesthesiology University of California, Davis—School of Medicine
7/87-9/91	Staff Anesthesiologist (Private Practice) American River Hospital Department of Anesthesiology Carmichael, CA
7/87-9/91	Assistant Clinical Professor (volunteer) Department of Anesthesiology University of California, Davis—School of Medicine

LICENSURE & CERTIFICATIONS:
State of California #G55662 (expires 7-31-2025)
State of Georgia #100252 (expires 7-31-2025)
DEA certificate BA0948870 (expires 6-30-2027)
Diplomate, National Board of Medical Examiners (1985)
Diplomate, American Board of Anesthesiology (1989; Life-time, not time limited)
Certificate of Recertification, American Board of Anesthesiology (1999, 2009)
Certified Yellow Belt, 2017

PROFESSIONAL SOCIETIES AND RECOGNITION:
American Society of Anesthesiologists 1987--present
California Society of Anesthesiologists 1987—present
Fellow of the American Society of Anesthesiologists 2018—present

ADVOCACY
ASA Grassroots Network (ASA Team 535) 2018
ASAPAC Donor—2018
FAER Donor—1999-2022

RESEARCH INTERESTS:

Mechanisms of anesthesia; factors influencing anesthetic requirements; OR efficiency

AWARDS AND HONORS

Dean's Mentoring Award, UC Davis School of Medicine, 2006
Associated Students of UC Davis "Excellence in Education Award" College of Biological Sciences, 2007
Associated Students of UC Davis "Excellence in Education Award" Outstanding Educator, 2007
Foundation for Anesthesia Education and Research, Mentor Academy, 2008
Phi Kappa Phi Honor Society, 2010

GRANTS

1. UC Davis Faculty Research Grant 1991-92—The effect of intrathecal aspirin on anesthetic requirements in rabbits, $2500
2. UC Davis Faculty Research Grant 1993-94—Validation of a preferentially anesthetized goat brain model, $1500
3. Foundation for Anesthesia Education and Research 1994—Determination of gross anatomic sites of anesthetic action, $25,000 ($25,000 matching departmental funds)
4. UC Davis Faculty Research Grant 1994-95—The effects of general anesthesia on cerebral blood flow patterns as assessed by functional magnetic resonance imaging, $1500
5. UC Davis Faculty Research Grant 1996-97—The effect of differential isoflurane delivery to brain and spinal cord on inhibitory and excitatory output from the brain, $10,000
6. Foundation for Anesthesia Education and Research 1997-99—The effect of differential isoflurane delivery to brain and spinal cord on inhibitory and excitatory output from the brain, $70,000 ($70,000 matching departmental funds)
7. NIH R01 GM57970 Brain and Spinal Cord Contributions to Anesthetic Action 8/98-4/02 (Priority Score 120, Percentile 1.0). Total costs $713,026
8. NIH R01 GM61283 Anesthetic Effects on Sensorimotor Integration 2/01-2/06 (Priority Score 194, Percentile 16.9). Total costs $672,791
9. U.C. Davis Faculty Research Grant. Indirect effect of isoflurane and lidocaine on EEG activation. 7/1/01-6/30/02, $4,000
10. NIH R01 GM57970-4A1 Brain and Spinal Cord Contributions to Anesthetic Action 4/02-12/05 (Priority Score 197, Percentile 20). Total costs $1,284,689
11. NIH 3R01GM057970-05S1 Brain and Spinal Cord Contributions to Anesthetic Action. Minority Supplement grant. 7/03-7/04. Total costs $55,932
12. NIH P01 GM47818 Anesthetic Effects on Spinal Nociceptive Processing 8/04-7/09 (Priority Score 185). Total costs $804,325
13. NIH R01 GM61283A1 Anesthetic Effects on Sensorimotor Integration 12/05-12/9 (Priority Score 158, Percentile 9). Total costs $748,432

TEACHING

Post-Graduate:

1. Resident lectures on neuroanesthesia, anesthetic mechanisms, malignant hyperthermia, neuromuscular blocking drugs, volatile anesthetics, anesthesia research. 1991-2019
2. Anesthesiology Department Journal Club 2013-2016
3. UCSF Changing Practice of Anesthesia—Faculty. September 2014: Peri-operative Medicine and Healthcare Reform: Challenges and Opportunities for Anesthesiology

Graduate:

Guest lecturer for NPB 219 (E. Carstens, Instructor). 1998-2003

Guest lecturer for NPB 112 (E. Carstens, Instructor). 2001-2008

Guest lecturer for first year medical students—pain physiology 2002-2003

Facilitator, Application of Medical Principles 2002-2008

Guest Lecturer, 210B (Systemic Physiology) January 2006

Instructor of Record, Applied Physiology and Pharmacology 2007, 2008

Undergraduate:

NPB 10—Elementary Human Physiology (4 units). 2001-2009

Freshman Seminar: The Supreme Court and You. (2 units) 1998-2010

Human Physiology (Los Medanos College) 2020

Biology of Health (Los Medanos College) 2020-22

Epidemiology (Holy Names University) 2020

MENTORED STUDENTS, RESIDENTS AND POST-DOCTORAL SCHOLARS

1.	Kevin Schwartz, M.D.	Resident	1993
2.	Michael Borges, M.D	Resident	1994
3.	Agi Melton, M.D.	Resident	1994
4.	Etsuo Tabo, M.D.	Post-Doctoral Scholar	1997
5.	Steven Jinks	Graduate Student	1998-2001
6.	Chris Simons	Graduate Student	1998
7.	Xiao Wei Wang, M.D.	Post-Doctoral Scholar	1999
8.	Xiaoguang Chen, M.D.	Post-Doctoral Scholar	2000
9.	Makoto Sudo, M.D.	Post-Doctoral Scholar	2000
10.	Satoko Sudo, M.D.	Post-Doctoral Scholar	2000
11.	Alison Fitzgerald	Undergraduate Student	2000-2001
12.	Andrew Hall	Undergraduate Student	2001

13. John Martin, M.D.	Resident	2001
14. Steve Jinks, PhD.	Post-Doctoral Scholar	2001-2004
15. Jason Cuellar, BS	Graduate Student	2003-2004
16. Linda Barter, MsVM	Graduate Student	2004-2007
17. Mashawn Orth	Graduate Student	2004-2005
18. Carmen Dominguez, MD	Assistant Professor	2003-2005
19. Lauire Mark	Undergraduate Student	2005-2006
20. Matthew LeDuc	Medical Student	2005
21. Toshi Mitsuyo, M.D.	Post-Doctoral Scholar	2004-2005
22. Kevin Ng, M.D.	Resident	2005-2006
23. JongBun Kim, M.D.	Post-Doctoral Scholar	2006
24. Sean Shargh	Undergraduate Student	2006-2007
25. Aubrey Yao, M.D.	Resident	2006-2007
26. Alana Sulger	Undergraduate Student	2006-2007
27. Gudrun Kungys, M.D.	Resident	2007-2008
28. Jason Talavera	Medical student	2007
29. Onkar Judge	Medical student	2008
30. Andrew Cunningham	Undergraduate Student	2008
31. Lauren Boudewyn	Undergraduate Student	2008
32. Austin Kim	Undergraduate Student	2008
33. Jason Andrada	Graduate Student	2009-2010
34. Jun Ye	Graduate Student	2014-2015
35. Reihaneh Forghany	Resident/Faculty	2018-2021

SPECIAL ACTIVITIES:

Staff Anesthesiologist, American River Hospital, 1987-1992

Medical Advisor, CMT International (Charcot-Marie-Tooth), 1991-2000

Director, Case Conferences, Department of Anesthesiology, April-June, 1992

Proctor, Medical Board of California, 1992

Staff Membership, Sutter Davis Hospital, Davis, CA, 1992-1995

Consultant, Malignant Hyperthermia Hotline, Malignant Hyperthermia Association of the United States (MHAUS), 1992-2002

Associate, UC Davis Diagnostic Malignant Hyperthermia Laboratory, 1992-2010

Member, Subcommittee on Experimental Neuroscience and Biochemistry, American Society of Anesthesiologists, 1996

Finance and Executive Committees, UC Davis Department of Anesthesiology, 1996-2002

Quality Assurance Committee, U.C. Davis Department of Anesthesiology, 1998-2004

Course Director, Annual U.C. Davis Anesthesiology Update (CME meeting), 1996-2003
California Society of Anesthesiologists: Educational Programs Committee, 1998-2000

Coordinator, Grand Rounds, Department of Anesthesiology, 1996

Professional Billing Workgroup, U.C. Davis, 1996-98

Question Writer, American Board of Anesthesiology, 1998-2001

Member, UC Davis Animal Care Committee, 2000-2003

Member, UC Davis School of Medicine Personnel Committee, 2003—2007; Chair 2007

Member, UCD Committee on Academic Personnel (Appellate Sub-committee) 2009-11

Management Advisory Committee, Department of Anesthesiology, 2007

Ad Hoc Reviewer for *Anesthesiology, Hospital Topics, Journal of Clinical Anesthesia, Journal of Comparative Neurology, Regional Anesthesia and Pain Medicine, Pain, Brain Research, Journal of Neuroscience, Anesthesia and Analgesia, British Journal of Anaesthesia, Neuroscience, Cephalgia, Neuroscience Letters, Journal of Chromatography, Basic & Clinical Pharmacology & Toxicology, Therapeutics and Clinical Risk Management.*

Member, VA Merit Review Subcommittee, Alcohol and Drug Dependence, 2002-2005

Editor, American Board of Anesthesiology/ American Society of Anesthesiologists In-Training Examination 2003-2008

Associate Editor, *Anesthesiology* 2005—2011

Faculty Executive Committee, School of Medicine 2009-2010

Chair, Faculty Executive Committee, School of Medicine 2010-2011

Member of various hospital committees 2011-2016: Medical Staff Executive Committee, Quality Safety Committee, OR Committee, Surgical Services Steering Committee, Hospital Billing Group

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EDITED BOOKS

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PUBLICATIONS

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2. Antognini JF and ND Kien. Cardiopulmonary bypass does not alter canine enflurane requirements. Anesthesiology 1992; 76:953-957.

3. Antognini JF. Intrathecal acetylsalicylic acid and indomethacin are not analgesic for a supramaximal stimulus. Anesthesia and Analgesia 1993; 76:1079-1082.

4. Antognini JF. Hypothermia eliminates isoflurane requirements at 20°C. Anesthesiology 1993; 78:1152-1156.

5. Antognini JF and GA Gronert. Succinylcholine causes profound hyperkalemia in hemorrhagic, acidotic rabbits. Anesthesia and Analgesia 1993; 77:585-588.

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7. Antognini JF and K Schwartz. Exaggerated anesthetic requirements in the preferentially anesthetized brain. Anesthesiology 1993; 79:1244-1249.

8. Antognini JF and PH Eisele. Anesthetic potency and cardiopulmonary effects of enflurane, halothane, and isoflurane in goats. Laboratory Animal Science 1993; 43(6):607-610.

9. Antognini JF. Splanchnic release of potassium after hemorrhage and succinylcholine in rabbits. Anesthesia and Analgesia 1994; 78:687-690.

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11. Antognini JF and ND Kien. A method for preferential delivery of volatile anesthetics to the *in situ* goat brain. Anesthesiology 1994; 80:1148-1154.

12. Antognini JF, BK Lewis and JA Reitan. Hypothermia minimally decreases nitrous oxide anesthetic requirements. Anesthesia and Analgesia 1994; 79:980-982.

13. Borges M and JF Antognini. Does the brain influence somatic responses to noxious stimuli during isoflurane anesthesia? Anesthesiology 1994; 81:1511-1515.

14. Antognini JF and ND Kien. Potency (minimum alveolar anesthetic concentration) of isoflurane is independent of peripheral anesthetic effects. Anesthesia and Analgesia 1995; 81:69-72.

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delivery of isoflurane to head and torso on lumbar dorsal horn activity. Anesthesiology 1998; 88:1055-61

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